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 NEWS  8  NOV 21  CAS patent coverage to include exemplified prophetic
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                  availability of new fully-indexed citations
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L2   ANSWER 1 OF 6     MEDLINE on STN                    DUPLICATE 1
AN   2008468659      MEDLINE
DN   PubMed ID: 18621683
TI   Respiratory syncytial virus uses a Vps4-independent budding
     mechanism controlled by Rab11-FIP2.
AU   Utley Thomas J; Ducharme Nicole A; Varthakavi Vasundhara; Shepherd Bryan
     E; Santangelo Philip J; Lindquist Michael E; Goldenring James R; Crowe
     James E Jr
CS   Department of Microbiology and Immunology, Vanderbilt University Medical
     Center, Vanderbilt University, Nashville, TN 37232, USA.
NC   DK48370 (United States NIDDK)
     T32 GM08554 (United States NIGMS)
SO   Proceedings of the National Academy of Sciences of the United States of
     America, (2008 Jul 22) Vol. 105, No. 29, pp. 10209-14.  Electronic
     Publication: 2008-07-09.
     Journal code: 7505876. E-ISSN: 1091-6490.
CY   United States
DT   Journal; Article; (JOURNAL ARTICLE)
     (RESEARCH SUPPORT, N.I.H., EXTRAMURAL)
     (RESEARCH SUPPORT, NON-U.S. GOV'T)
LA   English
FS   Priority Journals
EM   200808
ED   Entered STN: 24 Jul 2008
     Last Updated on STN: 20 Aug 2008
     Entered Medline: 19 Aug 2008
AB   Respiratory syncytial virus (RSV) infects polarized epithelia, which have
     tightly regulated trafficking because of the separation and maintenance of
     the apical and basolateral membranes.  Previously we established a link
     between the apical recycling endosome (ARE) and the assembly of RSV.  The
     current studies tested the role of a major ARE-associated protein,
     Rab11 family interacting protein 2 (FIP2) in the virus
     life cycle.  A dominant-negative form of FIP2 lacking its N-terminal C2
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domain reduced the supernatant-associated RSV titer 1,000-fold and also caused the cell-associated virus titer to increase. These data suggested that the FIP2 C2 mutant caused a failure at the final budding step in the virus life cycle. Additionally, truncation of the Rab-binding domain from FIP2 caused its accumulation into mature filamentous virions. RSV budding was independent of the ESCRT machinery, the only well-defined budding mechanism for enveloped RNA viruses. Therefore, RSV uses a virus budding mechanism that is controlled by FIP2.

L2 ANSWER 2 OF 6 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2008:825120 CAPLUS
TI Respiratory syncytial virus uses a Vps4-independent budding mechanism controlled by Rab11-FIP2
AU Utley, Thomas J.; Ducharme, Nicole A.; Varthakavi, Vasundhara; Shepherd, Bryan E.; Santangelo, Philip J.; Lindquist, Michael E.; Goldenring, James R.; Crowe, James E., Jr.
CS Departments of Microbiology and Immunology, Vanderbilt University Medical Center, Vanderbilt University, Nashville, TN, 37232, USA
SO Proceedings of the National Academy of Sciences of the United States of America, Early Edition (2008), (July 9 2008), 1-6, 6 pp.
CODEN: PNASC8
URL: http://www.pnas.org/cgi/reprint/0712144105v1
PB National Academy of Sciences
DT Journal; (online computer file)
LA English
AB Respiratory syncytial virus (RSV) infects polarized epithelia, which have tightly regulated trafficking because of the separation and maintenance of the apical and basolateral membranes. Previously we established a link between the apical recycling endosome (ARE) and the assembly of RSV. The current studies tested the role of a major ARE-associated protein, Rab11 family interacting protein 2 (FIP2) in the virus life cycle. A dominant-neg. form of FIP2 lacking its N-terminal C2 domain reduced the supernatant-associated RSV titer 1,000-fold and also caused the cell-associated virus titer to increase. These data suggested that the FIP2 C2 mutant caused a failure at the final budding step in the virus life cycle. Addnl., truncation of the Rab-binding domain from FIP2 caused its accumulation into mature filamentous virions. RSV budding was independent of the ESCRT machinery, the only well-defined budding mechanism for enveloped RNA viruses. Therefore, RSV uses a virus budding mechanism that is controlled by FIP2.

L2 ANSWER 3 OF 6 MEDLINE on STN DUPLICATE 2
AN 2006205838 MEDLINE
DN PubMed ID: 16611921
TI Mouse polyomavirus enters early endosomes, requires their acidic pH for productive infection, and meets transferrin cargo in Rab11-positive endosomes.
AU Liebl David; Difato Francesco; Hornikova Lenka; Mannova Petra; Stokrova Jitka; Forstova Jitka
CS Department of Genetics and Microbiology, Faculty of Medicine, Charles University in Prague, Vinicna 5, 128 44 Prague 2, Czech Republic.
SO Journal of virology, (2006 May) Vol. 80, No. 9, pp. 4610-22.
Journal code: 0113724. ISSN: 0022-538X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LA English
FS Priority Journals
EM 200605
ED Entered STN: 14 Apr 2006
Last Updated on STN: 23 May 2006

Entered Medline: 22 May 2006
AB Mouse polyomavirus (PyV) virions enter cells by internalization into smooth monopinocytic vesicles, which fuse under the cell membrane with larger endosomes. Caveolin-1 was detected on monopinocytic vesicles carrying PyV particles in mouse fibroblasts and epithelial cells (33). Here, we show that PyV can be efficiently internalized by Jurkat cells, which do not express caveolin-1 and lack caveolae, and that overexpression of a caveolin-1 dominant-negative mutant in mouse epithelial cells does not prevent their productive infection. Strong colocalization of VP1 with early endosome antigen 1 (EEA1) and of EEA1 with caveolin-1 in mouse fibroblasts and epithelial cells suggests that the monopinocytic vesicles carrying the virus (and vesicles containing caveolin-1) fuse with EEA1-positive early endosomes. In contrast to SV40, PyV infection is dependent on the acidic pH of endosomes. Bafilomycin A1 abolished PyV infection, and an increase in endosomal pH by NH4Cl markedly reduced its efficiency when drugs were applied during virion transport towards the cell nucleus. The block of acidification resulted in the retention of a fraction of virions in early endosomes. To monitor further trafficking of PyV, we used fluorescent resonance energy transfer (FRET) to determine mutual localization of PyV VP1 with transferrin and Rab11 GTPase at a 2- to 10-nm resolution. Positive FRET between PyV VP1 and transferrin cargo and between PyV VP1 and Rab11 suggests that during later times postinfection (1.5 to 3 h), the virus meets up with transferrin in the Rab11-positive recycling endosome. These results point to a convergence of the virus and the cargo internalized by different pathways in common transitional compartments.

L2 ANSWER 4 OF 6 MEDLINE on STN DUPLICATE 3
AN 2004559082 MEDLINE
DN PubMed ID: 15339909
TI Ganglioside GD3 traffics from the trans-Golgi network to plasma membrane by a Rab11-independent and brefeldin A-insensitive exocytic pathway.
AU Crespo Pilar Maria; Iglesias-Bartolome Ramiro; Daniotti Jose Luis
CS Centro de Investigaciones en Quimica Biologica de Cordoba, CIQUIBIC (UNC-CONICET), Departamento de Quimica Biologica, Facultad de Ciencias Quimicas, Universidad Nacional de Cordoba, 5000 Cordoba, Argentina.
SO The Journal of biological chemistry, (2004 Nov 12) Vol. 279, No. 46, pp. 47610-8. Electronic Publication: 2004-08-31.
Journal code: 2985121R. ISSN: 0021-9258.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LA English
FS Priority Journals
EM 200501
ED Entered STN: 9 Nov 2004
Last Updated on STN: 22 Jan 2005
Entered Medline: 21 Jan 2005
AB Gangliosides, complex glycosphingolipids containing sialic acids, have been found to reside in glycosphingolipid-enriched microdomains (GEM) at the plasma membrane. They are synthesized in the lumen of the Golgi complex and appear unable to translocate from the lumenal toward the cytosolic surface of Golgi membrane to access the monomeric lipid transport. As a consequence, they can only leave the Golgi complex via the lumenal surface of transport vesicles. In this work we analyzed the exocytic transport of the disialo ganglioside GD3 from trans-Golgi network (TGN) to plasma membrane in CHO-K1 cells by immunodetection of endogenously synthesized GD3. We found that ganglioside GD3, unlike another luminal membrane-bounded lipid (glycosylphosphatidylinositol-anchored protein), did not partition into GEM domains in the Golgi complex and trafficked from TGN to plasma

membrane by a brefeldin A-insensitive exocytic pathway. Moreover, a dominant negative form of Rab11, which prevents exit of vesicular stomatitis virus glycoprotein from the Golgi complex, did not influence the capacity of GD3 to reach the cell surface. Our results strongly support the notion that most ganglioside GD3 traffics from the TGN to the plasma membrane by a non-conventional vesicular pathway where lateral membrane segregation of vesicular stomatitis virus glycoprotein (non-GEM resident) and glycosylphosphatidylinositol-anchored proteins (GEM resident) from GD3 is required before exiting TGN.

L2 ANSWER 5 OF 6 MEDLINE on STN DUPLICATE 4
AN 2003019210 MEDLINE
DN PubMed ID: 12525601
TI Mouse polyomavirus utilizes recycling endosomes for a traffic pathway independent of COPI vesicle transport.
AU Mannova Petra; Forstova Jitka
CS Department of Genetics and Microbiology, Charles University in Prague, 128 44 Prague 2, Czech Republic.
SO Journal of virology, (2003 Feb) Vol. 77, No. 3, pp. 1672-81.
Journal code: 0113724. ISSN: 0022-538X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LA English
FS Priority Journals
EM 200302
ED Entered STN: 15 Jan 2003
Last Updated on STN: 12 Feb 2003
Entered Medline: 11 Feb 2003
AB Mouse polyomavirus enters host cells internalized, similar to simian virus 40 (SV40), in smooth monopinocytic vesicles, the movement of which is associated with transient actin disorganization. The major capsid protein (VP1) of the incoming polyomavirus accumulates on membranes around the cell nucleus. Here we show that unlike SV40, mouse polyomavirus infection is not substantially inhibited by brefeldin A, and colocalization of VP1 with beta-COP during early stages of polyomavirus infection in mouse fibroblasts was observed only rarely. Thus, these viruses obviously use different traffic routes from the plasma membrane toward the cell nucleus. At approximately 3 h postinfection, a part of VP1 colocalized with the endoplasmic reticulum marker BiP, and a subpopulation of virus was found in perinuclear areas associated with Rab11 GTPase and colocalized with transferrin, a marker of recycling endosomes. Earlier postinfection, a minor subpopulation of virions was found to be associated with Rab5, known to be connected with early endosomes, but the cell entry of virus was slower than that of transferrin or cholera toxin B-fragment. Neither Rab7, a marker of late endosomes, nor LAMP-2 lysosomal glycoprotein was found to colocalize with polyomavirus. In situ hybridization with polyomavirus genome-specific fluorescent probes clearly demonstrated that, regardless of the multiplicity of infection, only a few virions delivered their genomic DNA into the cell nucleus, while the majority of viral genomes (and VP1) moved back from the proximity of the nucleus to the cytosol, apparently for their degradation.

L2 ANSWER 6 OF 6 CAPLUS COPYRIGHT 2008 ACS on STN
AN 1998:725940 CAPLUS
DN 130:79126
TI Rab11 is required for trans-Golgi network-to-plasma membrane transport and a preferential target for GDP dissociation inhibitor
AU Chen, Wei; Feng, Yan; Chen, Dayue; Wandinger-Ness, Angela
CS Department of Biochemistry, Molecular Biology, and Cell Biology, Northwestern University, Evanston, IL, 60208-3500, USA

SO Molecular Biology of the Cell (1998), 9(11), 3241-3257
CODEN: MBCEEV; ISSN: 1059-1524
PB American Society for Cell Biology
DT Journal
LA English
AB The rab11 GTPase has been localized to both the Golgi and recycling endosomes; however, its Golgi-associated function has remained obscure. In this study, rab11 function in exocytic transport was analyzed by using two independent means to perturb its activity. First, expression of the dominant interfering rab11S25N mutant protein led to a significant inhibition of the cell surface transport of vesicular stomatitis virus (VSV) G protein and caused VSV G protein to accumulate in the Golgi. On the other hand, the expression of wild-type rab11 or the activating rab11Q70L mutant had no adverse effect on VSV G transport. Next, the membrane association of rab11, which is crucial for its function, was perturbed by modest increases in GDP dissociation inhibitor (GDI) levels. This led to selective inhibition of the trans-Golgi network to cell surface delivery, whereas endoplasmic reticulum-to-Golgi and intra-Golgi transport were largely unaffected. The transport inhibition was reversed specifically by coexpression of wild-type rab11 with GDI. Under the same conditions two other exocytic rab proteins, rab2 and rab8, remained membrane bound, and the transport steps regulated by these rab proteins were unaffected. Neither mutant rab11S25N nor GDI overexpression had any impact on the cell surface delivery of influenza hemagglutinin. These data show that functional rab11 is critical for the export of a basolateral marker but not an apical marker from the trans-Golgi network and pinpoint rab11 as a sensitive target for inhibition by excess GDI.
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